

V. CLAIMS

What is claimed is:

1. A method for inhibiting cancer cell proliferation comprising administering a NF- κ B inhibitor to a subject, wherein the NF- κ B inhibitor causes an NF κ B inhibition, wherein the subject has cancer cells which are proliferating, wherein the cancer cells are not myeloma cells.
- 2 A method of promoting cancer cell apoptosis comprising administering a NF- κ B inhibitor to a subject, wherein the NF- κ B inhibitor causes an NF- κ B inhibition, wherein the subject has cancer cells, wherein the cancer cells are not myeloma.
3. A method of inhibiting readhesion of cancer cells to a surface comprising administering a NF- κ B inhibitor to a subject, wherein the NF- κ B inhibitor causes an NF- κ B inhibition, wherein the subject has cancer cells.
4. A method of inhibiting metastasis of cancer cells comprising administering a NF- κ B inhibitor to a subject, wherein the NF- κ B inhibitor causes an NF- κ B inhibition, wherein the subject has cancer cells.
5. The method of claim 4, wherein the NF- κ B inhibitor inhibits intraabdominal metastasis.
6. The method of claim 4, wherein the NF- κ B inhibitor inhibits hepatic, parietal or peritoneal metastasis.
7. A method of inhibiting tumorigenesis comprising administering a NF- κ B inhibitor to a subject, wherein the NF- κ B inhibitor causes an NF- κ B inhibition, wherein the subject has cancer cells.
8. The method of any one of claims 1-4, wherein the cancer is an abdominal cancer, hepatic cancer, peritoneal cancer, parietal cancer, rectal cancer, stomach cancer, or colon cancer.
9. The method of any one of claims 1-4 or 7, wherein the cancer cells utilize NF- κ B for mitogenesis.
10. The method of any one of claims 1-4 or 7, wherein the cancer cells utilize NF- κ B for readhesion
11. The method of any one of claims 1-4 or 7, wherein the cancer cell comprises an

APC mutation.

12. The method of any one of claims 1-4 or 7, wherein the cancer cell does not contain an activating mutation on β -catenin.

13. The method of any one of claims 1-4 or 7, wherein the cancer cell expresses the COX2 gene.

14. The method of claim 12, wherein the cancer cell overexpresses the COX2 gene.

15. The method of claims 1-4, or 7, wherein the cancer cell does not express the COX2 gene.

16. The method of any one of claims 1-4 or 7, wherein the cancer cell is related to a cancer cell line.

17. The method of claim 14, wherein the cancer cell line is a DLD-1 cell line or a HT-29 cell line.

18. The method of any one of claims 1-4 or 7, wherein the cancer cells are colon cancer cells.

19. The method of any one of claims 1-4 or 7, wherein the cancer cells are rectal cancer cells.

20. The method of any one of claims 1-4 or 7, wherein the cancer cells are not adenocarcinoma cells.

21. The method of claim 1, wherein inhibiting cancer cell proliferation is independent of TNF α activated apoptosis.

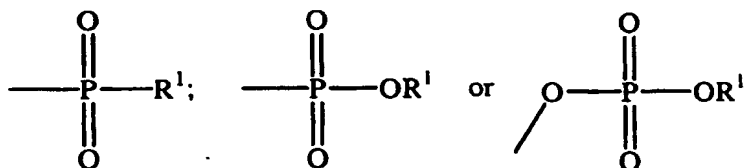
22. The method of claim 2, wherein promoting cancer cell apoptosis is independent of TNF α activated apoptosis.

23. The method of claim 3, wherein inhibiting readhesion of cancer cells to a surface is independent of TNF α activated apoptosis.

24. The method of claim 4, wherein inhibiting metastasis of cancer cells is independent of TNF α activated apoptosis.

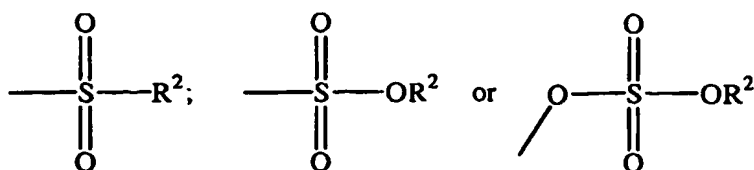
25. The method of claim 7, wherein inhibiting tumorigenesis is independent of TNF α activated apoptosis.

26. The method of any one of claims 1-4 or 7, wherein the NF- κ B inhibitor causes a decrease in the expression of anti-apoptotic proteins.
27. The method of any one of claims 1-4 or 7, wherein the NF- κ B inhibitor inhibits I κ B phosphorylation.
28. The method of any one of claims 1-4 or 7, wherein the NF- κ B inhibitor inhibits TNF α induced NF- κ B activation.
29. The method of any one of claims 1-4 or 7, wherein the NF- κ B inhibitor is an olefin.
30. The method of any one of claims 1-4 or 7, wherein the NF- κ B inhibitor is an olefin having at least one electron-withdrawing group.
31. The method of any one of claims 1-4 or 7, wherein the NF- κ B inhibitor is an olefin having at least two electron-withdrawing groups.
32. The method of claim 29, wherein the electron-withdrawing group comprises a cyano group, a sulfo-oxy group, a phospho-oxy group, a carboxyl group, a nitro group, a halogen, a halogenated alkyl group, an unsubstituted aromatic ring, or a substituted aromatic ring having at least one cyano group, sulfo-oxy group, phospho-oxy group, carboxyl group, hydroxyl group, amino group, ether group, halogenated alkyl group, halogen, or nitro group.
33. The method of claim 31, wherein the phospho-oxy group has the structure



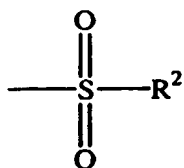
wherein R¹ is hydrogen, alkyl, halogenated alkyl, alkenyl, alkynyl, aralkyl, or substituted or unsubstituted aromatic. [N&R will define each of these terms in the specification.]

34. The method of claim 31, wherein the sulfo-oxy group has the structure



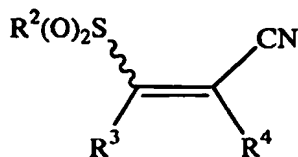
wherein R^2 is hydrogen, alkyl, halogenated alkyl, alkenyl, alkynyl, aralkyl, or substituted or unsubstituted aromatic.

35. The method of any one of claims 1-4 or 7, wherein the NF- κ B inhibitor is an olefin having a cyano group and a sulfo-oxy group having the structure



wherein R^2 is hydrogen, alkyl, halogenated alkyl, alkenyl, alkynyl, aralkyl, or substituted or unsubstituted aromatic.

36. The method of any one of claims 1-4 or 7, wherein the NF- κ B inhibitor has the structure



wherein R^2 , R^3 and R^4 are, independently, hydrogen, alkyl, halogenated alkyl, alkenyl, alkynyl, aralkyl, or substituted or unsubstituted aromatic, wherein the compound is the E- or Z-isomer.

37. The method of claim 35, wherein R^3 and R^4 are hydrogen.

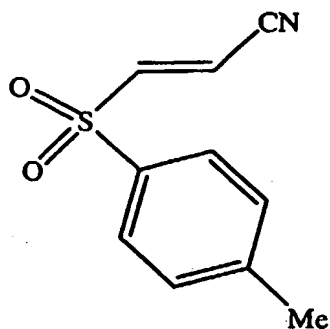
38. The method of claim 35, wherein R^2 is methyl, ethyl, propyl, isopropyl, butyl, isobutyl, tertiary butyl, substituted or unsubstituted phenyl, or benzyl.

39. The method of claim 35, wherein R^2 is a phenyl group having at least one alkyl

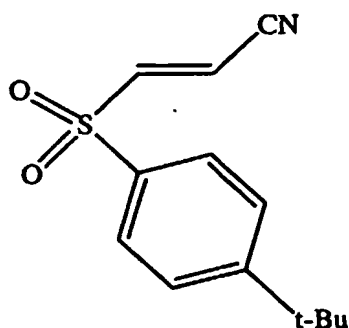
group.

40. The method of claim 35, wherein the compound is the E-isomer.

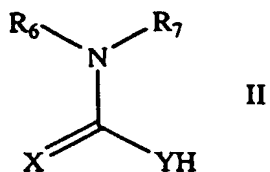
41. The method of any one of claims 1-4 or 7, wherein the NF-κB inhibitor has the structure



42. The method of any one of claims 1-4 or 7, wherein the NF-κB inhibitor has the structure



43. The method of any one of claims 1-4 or 7, wherein the NF-κB inhibitor has the structure



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wherein R_6 and R_7 are, independently, hydrogen, alkyl, alkenyl, alkynyl, aralkyl, or substituted or unsubstituted aromatic, or R_6 and R_7 together form a ring with the nitrogen atom, X and Y are, independently, oxygen or sulfur, or the pharmaceutically-acceptable salt, ester, or amide thereof.

44. The method of claim 43, wherein X and Y are sulfur, and R_6 and R_7 is $(CH_2)_4$.
45. The method of any one of claims 1-4 or 7, wherein the NF- κ B inhibitor comprises at least one amino acid residue.
46. The method of any one of claims 1-4 or 7, wherein the NF- κ B inhibitor has at least one leucine residue.
47. The method of any one of claims 1-4 or 7, wherein the NF- κ B inhibitor comprises three leucine residues.
48. The method of any one of claims 1-4 or 7, wherein the NF- κ B inhibitor is N-[(phenylmethoxy)carbonyl]-L-leucyl-N-[(1S)-1-formyl-3-methylbutyl]-L-leucinamide.
49. The method of claim 40, wherein the NF- κ B inhibitor is BAY-11-7082.
50. The method of claim 40, wherein the NF- κ B inhibitor is BAY-11-7085.
51. The method of any one of claims 1-4 or 7, wherein the NF- κ B inhibitor is MG-132.
52. The method of any one of claims 1-4 or 7, wherein the NF- κ B inhibitor is PDTC.
53. The method of any one of claims 1-4 or 7, wherein the NF- κ B inhibitor directly inhibits NF- κ B.
54. The method of any one of claims 1-4 or 7, wherein the NF- κ B inhibitor indirectly inhibits NF- κ B.
55. The method of 52, wherein the NF- κ B inhibitor inhibits expression of NF- κ B.
56. The method of 52, wherein the NF- κ B inhibitor inhibits translation of NF- κ B.
57. The method of any one of claims 1-4 or 7, wherein the NF- κ B inhibitor inhibits NF- κ B transport into the nucleus.
58. A method of inhibiting cancer cell proliferation in a subject, comprising testing

for an adenomatous polyposis coli (APC) gene mutation, and if the mutation is detected, administering an effective amount of an NF- κ B inhibitor to the subject.

59. The method of claim 58, wherein the NF- κ B inhibitor comprises BAY 11-7085.

60. The method of claim 58, wherein the NF- κ B inhibitor comprises BAY 11-7082.

61. A method of inhibiting cancer cell proliferation in a subject comprising testing the subject for COX2 expression, and if there was COX 2 expression, administering an NF- κ B inhibitor to the subject.

62. The method of claim 61, wherein the NF- κ B inhibitor comprises BAY 11-7085.

63. The method of claim 61, wherein the NF- κ B inhibitor comprises BAY 11-7082.

64. A method of inhibiting cancer cell proliferation in a subject comprising administering an NF- κ B inhibitor to the subject, wherein the subject has had a tumor resected.

65. The method of claim 64, wherein the NF- κ B inhibitor is administered prior to the resection.

66. The method of claim 64, wherein the NF- κ B inhibitor is administered prior to the resection.

67. The method of claim 64, wherein the NF- κ B inhibitor is administered within 10 days of the resection.

68. The method of claim 64, wherein the NF- κ B inhibitor is administered within 5 days of the resection.

69. The method of claim 64, wherein the NF- κ B inhibitor is administered within 1 days of the resection.

70. The method of claim 64, wherein the NF- κ B inhibitor is administered within 10 hours of the resection.

71. The method of claim 64, wherein the NF- κ B inhibitor is administered within 1 hour of the resection.

72. The method of claim 64, wherein the NF- κ B inhibitor is administered within 0.5 hours of the resection.